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16. ABSTRACT

This review puts into perspective the relationship of human morbidity to the occurrence of sulfur oxides and related particulates in the atmosphere. There is no question that such a relationship exists. But the many problems of terminology and technology make a precise and unequivocal interpretation of the data impossible. Most of the morbidity data come from epidemiologic studies; air pollution epidemiology is undeniably a multidisciplinary endeavor with all the problems inherent therein; its interpretation, when recognizing these problems, can provide a holistic view that is greater than the sum of its components. Any single study in air pollution epidemiology could lead equally qualified, honest individuals to widely divergent interpretations. The data must, therefore, be interpreted in a critical, but open-minded fashion that recognizes methodologic problems and leans toward qualitative conclusions rather than straining for rigid quantitative results. This evidence provides a basis into which can be inserted the more quantitative data from animal toxicology, in vitro metabolic studies, and clinical research that lead to the final judgmental process of developing regulatory policy.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
AIR POLLUTION SULFUR OXIDES PARTICLES EPIDEMIOLOGY		06.F

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EVIDENCE FOR THE INFLUENCE OF SULFUR OXIDES AND PARTICULATES ON MORBIDITY*

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THIS nation is being forced into an energy-production policy that may significantly increase emissions and atmospheric concentrations of atmospheric sulfur oxides and particulates unless relatively costly control measures are taken. We must attempt to assign relative importance to the effects observed to balance the cost of controlling pollutants against the cost in human disease, misery, and unhappiness if these pollutants are not controlled. These comparisons must be explicit and objective, with the rules for assessing costs versus benefits defined in advance.

A few cautionary statements are necessary. The relation between sulfur oxides and particulate air pollution and possible health effects is fraught with more difficulty than most areas of environmental medicine. Failure to recognize these difficulties at the outset would ill serve those who must eventually make the cost/benefit analyses. The uncertainties are great, and will lead some to conclude that the costs of control at a particular level are unbearable until the disease-related costs can be described with more precision. These same uncertainties will lead others to the opposite conclusion: that the risks of *not* controlling at a particular level are unacceptable until that health risk is assessed with equally greater precision.

The most difficult aspect of this topic is the absence of uniform terminology. What are particulates? What are sulfur oxides? Without a reasonable consensus on definition of the problem we cannot solve the second greatest difficulty—the development of adequate measurement

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technology. Assessment of human morbidity has its own inherent problems. But with sulfur oxides and particulate pollution, to what specifically do we relate that morbidity? Each study must be carefully scrutinized, and the temptation to draw specific conclusions must be resisted when data simply do not support them.

Interpretation of Air Quality Data

With these precautions in mind, there is a certain logic in examining sulfur oxides and particulates together. Sulfur oxides are the greatest single component of the total suspended particulate (TSP) mix, about 10-11%.[1] An even greater fraction of sulfur oxides concentrates in the smaller, more respirable part of the bimodal TSP distribution because these compounds form predominately from condensation reactions producing aerosols from gas-phase emissions.[2,3] Nitrates, which for the same reason tend to be concentrated in the small particle fraction, compose 2-3% of TSP,[1] and, when specific chemical analyses are not available, one cannot say with much certainty what fraction of the observed effect should be attributed to either major fraction of the more respirable portion of TSP.

To complicate the issue further, the second greatest fraction of TSP is what is referred to as benzene-soluble organics (BSO), about 6-7% of TSP. Because these aerosols also arise primarily from condensation reactions, they too tend to concentrate in the more respirable fraction of TSP. Except for iron and ammonium ions (both about 1-2%), all other constituents of TSP are orders of magnitude lower in concentration. Benzene-soluble organics are such a heterogenous mix that very little can be said about their contribution to morbidity in relation to TSP concentrations or other particulate levels in the atmosphere. However, it is reasonable that some fraction of the observed health effects be attributed to constituents of BSO with known toxic properties, including some probable carcinogens. Although geographical variations in mortality must be interpreted cautiously, this reasoning with respect to BSO is consistent with the observations of Stocks et al. and Wicken and Buck that lung-cancer mortality strongly correlated with smoke density in various areas of Great Britain.[4-9]

In discussing human morbidity related to sulfur oxides and particulates it must be recalled that these studies used various techniques to assess population exposure. Frequently, sulfur dioxide was the independent variable. I believe we should not infer a direct relation between any single atmospheric measurement and observed health effects. We know that the

specific composition of the sulfur oxides/particulate mix depends on a variety of factors, including emissions sources, meteorologic conditions, and terrain. Composition varies from place to place and time to time.

Yet some generalizations can be drawn from available data,[1] and for the purposes of discussion we can accept some of these generalizations rather than dwell on the problems I have discussed. It seems helpful to recognize that certain sulfur dioxide levels are likely to be accompanied by some amount of sulfur oxide aerosols. Studies that report only Smoke Shade, coefficient of haze (COH), or TSP measurements in areas where known emission inventories include sulfur compounds should probably attribute some of the observed health effects to sulfur oxide aerosols, even if these compounds were not specifically measured.

Interpretation of Morbidity Data

The earliest studies deal primarily with mortality, but also refer to morbidity, and these early anecdotal reports are substantially corroborated by subsequent studies. An association between noxious fumes or smoke and undesirable public health conditions is made even in the middle ages, but Firket made some quantitative observations in his discussion of the Meuse Valley episode of December 1930.[10-11] Eighteen years later, in October 1948, Donora, Pa., was blanketed by a thick fog reminiscent of the Meuse Valley episode. Again, morbidity and mortality were inescapably associated in the minds of those present with unusual atmospheric conditions and high levels of irritating pollutants in the air, but aerometric measurements were not made during the episode and retrospective epidemiologic analyses by Schrenk et al. could not separate the effects of specific pollutants.[12] Subsequent high levels of sulfur dioxide were found in Donora during inversions similar to but not so severe as that of October 1948. Thus, it seems likely that sulfur oxides contributed significantly to the excess morbidity and mortality of the Donora episode. Firket calculated that an episode similar to that of the Meuse Valley in London would cause some 3,200 deaths.[10] In 1952 such a disastrous episode did occur in London with 4,000 associated excess deaths. On this occasion SO_2 measurements were available and correlated with the excess mortality.[12] Analyses of subsequent, less severe episodes confirmed that elevated sulfur dioxide levels and smoke measurements correlated with excess mortality.[13,14]

Attempts to relate human health effects to rapidly changing environmen-

tal conditions suffer from serious methodologic difficulties. The size of the population studied must be substantial if unexplained excesses in morbidity or mortality are to be expected to emerge from normal secular variability. Episodes are, by definition, unpredictable, and, even when continuous air-monitoring data are available, the health studies must be done in retrospect. This is not a major problem for mortality studies where vital statistics and hospital records are relatively adequate, but retrospective assessment of morbidity has greater uncertainty.

Modern improvements in technology have enhanced our ability to extract useful information from episodes, and improved statistical approaches provide meaningful analyses of smaller populations. Greatly improved exposure assessment combines better air-measurement methods with atmospheric modeling. Better understanding of meteorologic trends permits more reasonable forecasting of air-pollution episodes.

Several air-monitoring stations of the Allegheny County Air Pollution Control Board reported as an example, increasing concentrations of particulate matter (COH) on November 18, 1975. Pollution levels continued to increase that night and the following morning and it was predicted that adverse meteorologic conditions would persist. About 10:00 A.M. on November 19, 1975 the Environmental Protection Agency's (EPA) Health Effects Research Laboratory in North Carolina was notified of a potential significant air-pollution episode in Allegheny County, Pa. Four hours later an investigative team of physicians, epidemiologists, air-monitoring specialists, and a meteorologist were enroute to Pittsburgh by Lear Jet. By 8:00 P.M. that evening an on-site evaluation of the situation in Allegheny County had been made and a study design prepared to test possible effects of high air-pollution levels on pulmonary function in children. On the afternoon of November 19, 1975 a specially equipped mobile air-pollution monitoring laboratory left North Carolina and was on site by noon the next day measuring sulfur dioxide, TSP, suspended sulfates, and suspended nitrates. Acquisition of pulmonary-function data from school children began on the morning of November 20, and was fully under way the next morning. Concurrently, plans for a mortality study of this episode were initiated.

This detailed description of an episode response emphasizes the necessity for rapid initiation of a multidisciplinary high technology approach if studies of this nature are to be successful. Four observations emerged from this experience:

1) Given the resources, all appropriate personnel and instrumentation can converge on the study site and initiate high-quality data acquisition within hours of an alert.

2) When spirometry data were analyzed using standard statistical techniques, no gross decrements in lung mechanical function were observed among children during initial phases of testing immediately following the episode, nor did lung function detectably improve with serial testing of the same children several days following the episode.[15]

3) Using more innovative statistical methods to identify "susceptible" segments of the schoolchild population, significant improvement in lung function occurred in these children following the episode.[16]

4) Again, carefully obtained vital statistics data, continuous exposure-assessment data, and more recently developed daily mortality models described a small but significant increase in mortality during this episode of high TSP, high sulfur dioxide levels, and high suspended sulfate levels.[17]

Thus, a range of observations based on air-pollution episodes has been presented. Those of the Meuse Valley, Donora, and London are convincing qualitatively, but little quantitative information can be extracted from them. The Pittsburgh episode of 1975 provided observations of relatively subtle specific changes, relatable to well-documented changes in air quality. These observations further depended on the ability to commit major research resources in an atmosphere reminiscent of a military operation. These opportunities do not occur often. The greatest value of the results of air-pollution episode studies probably lies in their providing well-thought-out hypotheses to be tested by carefully designed prospective epidemiologic studies, and by clinical studies to evaluate specific effects of specific atmospheric constituents in humans.

Much morbidity data on sulfur oxides and related particles comes from epidemiologic studies because methodologic difficulties until recently precluded much clinical research. It is impossible to assess truly chronic effects in clinical studies or to use any simulated exposures that have even a remote possibility of serious or irreversible effects on the subjects.

We have attempted to describe in general terms some of the background and problems in attempting to relate human disease to atmospheric concentrations of sulfur oxides and related particulates. Much of the basis for current standards depends on interpretation of the early air-pollution episodes.

The current United States ambient air-quality standard (AAQS) for sulfur oxides is 80 μg./m.^{3} SO_2 annual arithmetic mean and 365 μg./m.^{3} SO_2 maximum 24-hour concentration not to be exceeded more than once a year. The AAQS for TSP is 75 μg./m.^{3} annual geometric mean and 260 μg./m.^{3} 24-hour average. Both standards are largely based on the same massive, short-term population exposures which resulted in immediate, apparent human death and disease, exposures we have referred to as episodes. These standards are based on the respective Air Quality Criteria Documents.[18,19] Brief comments on the more important studies may provide a point of reference to discuss research done since these documents were published.

Brasser et al.[13] reviewed major episodes of air pollution in London and Rotterdam between 1952 and 1962. These studies indicate excess mortality when 24-hour mean levels of SO_2 exceed 500 μg./m.^{3} for a few days, and that hospital admission for respiratory illness and absenteeism increase whenever 24-hour mean levels of SO_2 reach 300 to 400 μg./m.^{3} for three to four consecutive days. Certain levels of particulate matter accompanied the observed excursions in SO_2, but from this report no portion of the observed health effects can be attributed to particulates. The same comment is appropriate with respect to the findings of Joosting[14] that significant correlations between SO_2 levels and death or disease occur with 24-hour mean SO_2 concentrations of 400 to 500 μg./m.^{3} in the presence of high levels of soot. Carnow et al.[20] reported increased illness among older people with more severe bronchitis when concurrent or preceding daily levels of SO_2 ranged from 119 to 249 μg./m.^{3} These findings were not observed in the patients younger than 55 years of age or in those with less severe bronchitis. During the study period TSP levels exceeded the 24-hour standard on about 15% of the days, so some portion of the increased illness could be attributed to elevations in particulate exposure as well as to SO_2.

Douglas and Waller[21] studied a large group of children for several years and observed increased lower respiratory illness among children who had lived in areas of higher pollution. No changes were found in upper respiratory disease incidence. Four categories of air-pollution levels were created for various areas of residence of the children, and air-pollution levels were calculated from known fuel consumption over the years. Actual atmospheric measurements, when possible, indicated that these categories were reasonably good when used as an index of SO_2 and smoke

levels, but some overlap in the categories for residential areas occurred, as might be expected. The increases in lower respiratory tract infections were observed for girls as well as boys in all socioeconomic classes, and appeared to persist until about 15 years of age. Areas of residence which showed this increased morbidity had annual mean SO_2 concentrations calculated to be 130 to 148 $\mu g./m.^3$ or higher and annual TSP levels calculated to be 91 to 138 $\mu g./m.^3$ or higher. Again, the relative contribution of SO_2 and TSP cannot be assessed in this study.

Colley et al.[22] were able to follow a large fraction of this cohort until age 20. They found no relation between respiratory symptoms, as reported on the standard British Medical Research Council questionnaire, and current levels of air pollution, nor were any relations between the symptoms and any socioeconomic or demographic factors identified. There was a highly significant relation, however, between symptom reporting and current smoking habits and previous history of childhood lower respiratory tract illness. One could conclude from this study that the previous excess of lower respiratory tract illness associated with childhood exposure to SO_2 and TSP was related to persistent excess respiratory symptoms when those children became adults, but that their current smoking habits obscured any relation to current air-pollution levels. An alternate interpretation is that symptoms from previous exposure persisted to prevent detection of continued deterioration of health related to current exposures.

Kiernan et al.[23] followed this same cohort for another five years, to age 25, with essentially the same findings of Colley et al.[22] Respiratory morbidity continued to be related to childhood lower respiratory infections in turn related to air-pollution levels experienced during childhood. Colley and Kiernan's studies did not figure in the promulgation of the current AAQS, but are discussed here rather than later because they are so elegant a sequel to the study of Douglas and Waller.

Lunn et al.[24] in 1963-1965 studied four areas of Sheffield, England, and found that both upper and lower respiratory illnesses were more frequent in young children living in the more polluted areas. These increases in morbidity were associated with a range in concentrations of both SO_2 and TSP, and this study largely corroborates those of Douglas and Waller. A distinct advantage of this study is the careful air-pollution measurements as compared to the estimates Douglas and Waller had to work with. The best data available during the period of this study show that mean daily figures for SO_2 ranged from 109 to 134 $\mu g./m.^3$ in the cleanest area. The

comparable values for TSP were 70 to 97 μg./m.^{3} in the dirtiest area, mean daily figures for SO_2 were 275 to 304 μg./m.^{3} and for TSP were 249 to 301 μg./m.^{3} Many parameters of respiratory illness showed a statistical gradation of effect between all four areas. Thus, excesses were not seen at levels of TSP below about 100 μg./m.^{3} accompanied by SO_2 below about 133 μg./m.^{3} Excesses began to appear in the next most polluted area at levels of TSP between 177 and 230 μg./m.^{3} accompanied by SO_2 between 181 and 194 μg./m.^{3}. It seems useful to compare these numbers to the AAQS of 75 μg./m.^{3} TSP and 80 μg./m.^{3} SO_2 (annual averages) to see the relatively small margin of safety between these values and the levels where effects began to appear in this study by Lunn et al.[24]

Lung-function evaluation conducted in this study ($FEV_{0.75}$ and FEV) demonstrated no direct relation to air-pollution levels, but was related to respiratory illness in the children which was, in turn, related to levels of air pollution. Lunn et al.[25] reported a follow-up study of 558 nine-year-old children who were first seen at age five years.[24] They also reported the results from 1,049 11-year-old children seen during that earlier study. The 11-year-olds had less respiratory illness than the five-year-olds seen at the same time, as one would expect, but a relation emerged for the 11-year-olds between respiratory illness morbidity and air pollution as categorized by area of residence. The original group of five-year-olds seen four years later at age nine had less respiratory illness than the 11-year-olds seen initially, and showed no difference in morbidity between areas of residence. These results are consistent and persuasive when compared to the substantial improvement in air quality in these residential areas during the four-year interval between the two studies.

Most of the air-pollution epidemiologic studies conducted in the United States since the publication of the air-quality criteria documents on sulfur oxides and particulate matter has been under the auspices of EPA. Much of this research was organized into a large-scale program known as the Community Health and Environmental Surveillance System (CHESS), and was the first comprehensive nationwide attempt at systematic surveillance of general populations exposed to varying air quality ever initiated anywhere. It was a large program, involving the full-time efforts of many people, which cost, during its five years of existence, several million dollars. Understandably a program of this magnitude attracted attention, scrutiny, and criticism. The first major publication, comprising a small fraction of all the CHESS data available, appeared in May 1974 and is

commonly referred to as the CHESS Monograph.[26] This monograph is not the only source of information on CHESS data, however.[27-62]

As a result of some of the controversy accompanying such a large program with so many publications in a relatively short time, a Congressional investigation was initiated. Prior to this investigation, EPA's philosophy of interpreting research data and applying it to the regulatory process had been carefully and eloquently presented at the National Academy of Sciences.[63] In that paper Dr. J.F. Finklea presented a detailed analysis of procedures to make "best case judgments" with respect to levels of air pollutants, including sulfur oxides and associated particulates, that are related to unacceptable risk of human morbidity.

The Congressional investigation of CHESS resulted in several reports [64-67] which identified many problems in air-pollution epidemiology. The investigative report[66] was particularly thorough and objective. The CHESS program shared many of the inherent problems of previous air-pollution epidemiology but was a pioneering effort that made a very significant contribution to our understanding of the health effects of sulfur oxides and particulates, especially at relatively low concentrations.

The earliest CHESS studies focused on chronic bronchitis among adults and lower respiratory disease among children through 12 years of age in four major geographic areas: the Salt Lake Basin in Utah, smelter communities in Idaho and Montana, the New York metropolitan area, and the Chicago metropolitan area. In each area, at least three separate communities were chosen to represent a gradient of exposure to sulfur oxides and particulates. EPA measurements of SO_2, TSP, and total suspended sulfates during the study period, historical data from state agencies, and emission inventories going back as far as 1940 were used to construct the best possible long-term exposure estimates.

In the Salt Lake Basin prevalence of chronic bronchitis among 7,635 adults was found to increase significantly among smokers and nonsmokers of both sexes after four to seven years exposure to elevated levels of SO_2 (about 90 to 95 μg./m.^{3}) and suspended sulfates (about 15 μg./m.^{3}) accompanied by relatively low levels of TSP (averaging about 60 μg./m.^{3}). The occurrence of lower respiratory disease in 9,000 preschool and elementary school children was 40 to 50% greater in children who lived for two years or more in the neighborhoods with higher levels of air pollution compared to those children residing in the neighborhoods with less pollution.

In the Idaho-Montana studies conducted around smelters, chronic bronchitis prevalence was again significantly greater, with two to three exposures to SO_2 levels ranging from 200 to 400 μg./m.^{3} and suspended sulfate levels ranging from 7 to 20 μg./m.^{3} in the presence of low levels of TSP. Nonsmokers in the communities with high exposures had chronic bronchitis rates two to three times greater than similarly nonoccupationally exposed nonsmokers in cleaner communities. Acute lower respiratory morbidity in children was significantly elevated after three or more years exposure to estimated annual averages of SO_2 of 177 μg./m.^{3} accompanied by low estimated annual averages of TSP (65 μg./m.^{3}) and estimated averages of suspended sulfates of about 7 μg./m.^{3}.

The Chicago-area studies differed somewhat in methodology from those conducted in Utah and the Rocky Mountain smelter communities. In Illinois military recruits were surveyed with respect to chronic bronchitis-prevalence rates which were compared to pollution levels in the communities where they had resided prior to induction into the military. Exposures lasting 12 years or more to annual sulfur dioxide levels ranging from about 90 to 200 μg./m.^{3}, TSP ranging from about 100 to 150 μg./m.^{3}, and suspended sulfates of about 14 μg./m.^{3} were associated with significant increases in chronic respiratory disease symptoms.

More than 2,500 family members of Chicago children in day-care centers were evaluated for acute respiratory illness in a prospective 10-month study. Acute respiratory illness occurred with significantly increased frequency in those individuals exposed to SO_2 levels ranging from 100 to 250 μg./m.^{3} than in those exposed to average annual levels of about 100 to 130 μg./m.^{3} The differences in TSP between the areas (135 to 165 μg./m.^{3} compared to about 120 μg./m.^{3}) was marginal. Annual suspended sulfate levels ranged from about 14 to 18 μg./m.^{3} during the period of the study, but there was little difference between communities.

In the New York studies, chronic respiratory symptoms were evaluated in 6,004 parents of elementary school children, and acute respiratory disease morbidity was assessed in the parents as well as their children. Exposures up to 20 years to SO_2 levels ranging from 140 to 400 μg./m.^{3} and suspended sulfate levels between 10 and 25 μg./m.^{3} were associated with marked increases in chronic bronchitis prevalence among smokers, nonsmokers, and exsmokers of both sexes 20 to 50 years of age.

Shorter term, more recent exposures to lower levels were associated with increased incidence, aggravated severity, or prolongation of chronic

respiratory symptoms. These shorter term exposures typically involved annual average SO_2 levels of 50 to 60 μg./m.^{3}, TSP levels ranging from 60 to 100 μg./m.^{3}, and average annual suspended sulfate levels of about 14 μg./m.^{3} The population composed of adults and children demonstrated a significant increase in frequency and severity of acute lower respiratory illness related to exposures of two to three years to annual average levels of SO_2 (255 to 320 μg./m.^{3}), TSP (95 to 125 μg./m.^{3}), and suspended sulfate (10 to 15 μg./m.^{3}).

In all these studies, appropriate corrections for demographic characteristics were made, and other common air pollutants were measured but did not contribute to the observed differences in morbidity.

It is not possible to take all the morbidity statistics with related estimates or measurements of air quality and arrive at a specific level of any pollutant below which we are confident no morbidity will occur. However, the consistency between these four CHESS studies and the consistency between the CHESS studies and those conducted elsewhere earlier is simply too striking to ignore. There remains no doubt that increased levels of sulfur oxides and associated particulates is related to increased morbidity in populations. Precisely what levels of which specific constituents of this sulfur oxides/particulate mix is related to what degree of morbidity with what degree of probability remains to be determined.

Experiments involving human subjects can contribute significantly to our understanding of the health effects of sulfur oxides, but only in narrowly defined areas. Sim and Pattle[68] and Frank and his co-workers[69-72] provide most of the work relating SO_2 alone and combined with sodium chloride aerosols to various aspects of respiratory uptake, deposition, and lung function. Related work has been done by Nadel et al., Tomono, Burton et al., and Amdur et al.,[75-80] which has aided greatly in identifying probable mechanisms of action on the respiratory tract using relatively high concentrations, and has paved the way for current research. Bushtueva[81,82] and her colleagues took a different important approach to define the level at which the presence of SO_2 or sulfur oxide aerosols can be detected using psychophysiologic methods, an approach not very actively pursued by others.

McJilton and Frank demonstrated synergism between SO_2 and aerosol mixtures in experimental animals, and are currently pursuing this work with human subjects.[83-85]

Future Advances

I earlier emphasized the two main probjems in assessing morbidity related to sulfur oxides and particulates: lack of uniform definitions and terminology and lack of adequate measurement methodology. My colleagues and I at EPA's Environmental Research Center in North Carolina made some specific recommendations.[86] Clearly, we cannot continue to measure TSP and SO_2 and expect to refine our assessment of morbidity related to airborne sulfur oxides and related particulates. Measurement of suspended sulfates and, in a few cases, suspended nitrates, has been useful. In an epidemiologic study now underway in St. Louis we measure SO_2 (with a flame photometric technique), TSP with a Hi Vol sampler, and particle sizes using an Anderson cascade impactor. We have in operation a continuous measurement of H_2SO_4, nitrogen dioxide, and ozone. The samples from both the Hi Vol and the Anderson sampler will be analyzed for total sulfates and nitrates. These measurements are being conducted at two outdoor and two indoor locations in an attempt to give us the best possible exposure assessment of our study population. This is an example of what we were doing in February 1978. But this is still not good enough.

A definition of "respirable" particulate (RSP) must be adopted and instrumentation deployed to identify the composition of RSP before significant advances can be made in toxicology and clinical studies. The relevance of epidemiology will be greatly enhanced by the addition of such measurements, and we believe the definition of RSP should be based on human anatomic and physiologic considerations. This will lead to the direct application of monitoring data to human morbidity data. Efficient multistage samplers have been developed, but are in limited use because of the relatively great cost of building and maintaining them. Increasing the number of fractions collected multiplies accordingly the expense of chemical analysis. We believe a reasonable compromise exists in the deployment of a two-stage size fractionating sampler with the upper cutoff at 15 μm. (aerodynamic equivalent diameter) and a second separation for particles 2 μm. and smaller. These numbers are based on anatomic and physiologic considerations and obviously represent a compromise. A network of such instruments adjacent to certain existing samplers of various design characteristics would enable us simultaneously to obtain new data of importance and to compare those data with data previously collected under a variety of

circumstances so that some comparisons and extrapolations would be possible.

Development of uniformity in definitions and exposure-assessment technology is not meant to preclude continued advancement in these areas of research, but to promote, indeed to permit, comparability of data, a "benchmark" must be established. This benchmark will be somewhat arbitrary and will not satisfy everyone. In fact, it may not satisfy anyone completely but it should partially satisfy enough of the people involved so that the objective of obtaining comparable data from multiple sources can be achieved.

Summary and Conclusions

I have tried to put into perspective the relation between human morbidity and sulfur oxides and related particulates in the atmosphere. Unquestionably, such a relation exists, but the problems of terminology and technology make precise and unequivocal interpretation of the data impossible. Most morbidity data come from epidemiologic studies. I do not believe epidemiology is a separate scientific discipline so much as an approach whereby researchers bring a variety of scientific tools to bear on public health problems. My opinion is that air-pollution epidemiology is a multidisciplinary endeavor with all the problems inherent therein. I believe a portion of our problem lies in our lack of recognition of specific difficulties involved in the design, management, and interpretation of air-pollution epidemiology. This endeavor involves a wide spectrum of activity with marked geographic and temporal dispersion. These studies involve large numbers of people from highly varied technical backgrounds—engineers, instrumentation specialists, meteorologists, data processors, statisticians, questionnaire specialists, epidemiologists, and physicians, all of whose activities must be meshed. As the number of people involved increases, the number of possible interactions increases exponentially. These people work within different paradigms, have different perspectives, obtain gratification differently, and expect recognition in various ways. This leads to conflict. Interpretation of air-pollution epidemiology, recognizing these factors, can provide a total view greater than the sum of its components. We who manage, conduct, and interpret air-pollution epidemiology studies intuitively appreciate the problems to which I allude, but I believe the quality of our endeavor will improve if we identify, analyze, and cope with these factors more explicitly.[87]

For these reasons I do not believe that air-pollution epidemiology can lead us very close to a quantitative assessment of the human morbidity associated with long-term exposures to sulfur oxides and particulate pollution. Indeed, I am unaware of any single study in air-pollution epidemiology, the results of which could not lead equally qualified, honest individuals to widely divergent interpretations. These data must, therefore, be interpreted in a critical but open-minded fashion that recognizes methodologic problems and leans toward qualitative conclusions rather than straining for rigid quantitative results. For these reasons I attempt no quantitative summary of the studies I have discussed. We must instead view the body of literature and decide on the preponderance of evidence. This basis can augment more quantitative data from animal toxicology, *in vitro* metabolic studies, and clinical research that lead to the final judgmental process of developing regulatory policy.[88]

Finally, we must strive for objectivity rather than advocacy. I have participated in and observed the adversary approach in the translation of human health information into regulatory policy and find it inefficient if not detrimental. I agree with Robert Lubar that "As for the environmentalists, they've been so successful in making their points that they have become a part of the establishment. They're no longer counterculture. Now they have to act with responsibility."[89] Extremist interpretation of the data in either direction is damaging. If environmentalists are part of the establishment and are to act responsibly, they deserve to have their industrial counterparts act with equal responsibility to arrive at reasonable interpretations of the pollution-related morbidity data we have and that which we shall continue to obtain.

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